no region exists where the oxygen pressure is sufficiently low to permit spore germination. However, if one injects tetanus spores into the blood of tumor-bearing mice, the mice sicken with tetanus, because the oxygen pressure in the tumors can be so low that the spores can germinate. These experiments demonstrate in a unique way the anaerobiosis of cancer cells and the non-anaerobiosis of normal cells, in particular the non-anaerobiosis of growing embryos.

The Fermentation of Morris Hepatomas

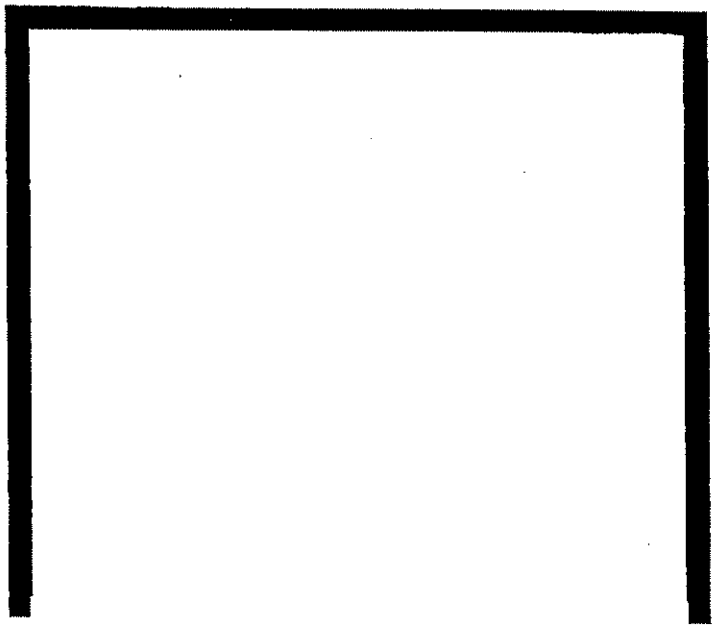
A second type of experimentation demonstrates a quantitative connection between fermentation of tumors and growth rate of tumors.

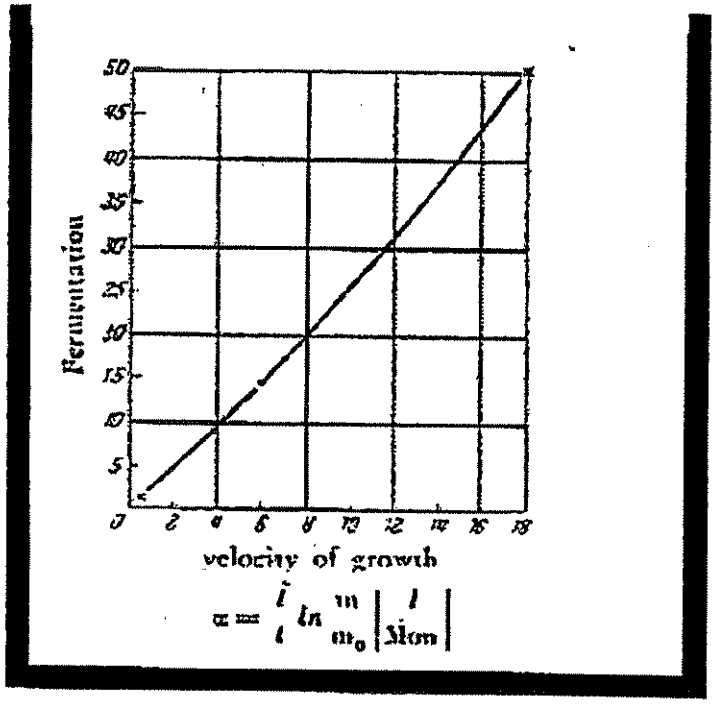
If one injects rats with cancer-inducing substances of different activities, one can create, as HAROLD MORRIS of the National Cancer Institute in Bethesda has found, liver cancers (hepatomas) of very different degrees of malignancy. Thus, one strain of tumor may double its mass in three days, another strain may require 30 days. Recently DEAN BURK and MARK WOODS 3), also of the National Cancer Institute, measured the in vitro rates of anaerobic fermentation in different lines of these hepatomas, and obtained a curve (Fig. 1) that shows a quantitative relationship between fermentation and growth rate, and therefore between fermentation and malignancy, in these various tumor strains. The fermentation increases with the malignancy, and indeed the fermentation increases even faster than the malignancy.

Special interest attaches to the fermentation of the most slowly growing hepatomas, because several investigators in the United States believed that they had found *) that such tumors had no fermentation; that is that anaerobiosis cannot be the prime cause of cancer.

*) For example see C. H. BÖHRINGER SON, Ingelheim am Rhein, the factory Work-Journal "Das Medizinische Prisma" , Vol. 13, 1963. Here a lecture of VAN POTTER (Madison, Wisconsin) is reprinted where owing to the slow-growing Morris-tumors anaerobiosis as prime cause of cancer is rejected and the lack of "intracellular feeding back" is claimed to be the real cause of cancer.

Fig. 1. Velocity of growth and fermentation of the Morris-Hepatomas, according to DEAN BURK and MARK WOODS





DEAN BURK and MARK WOODS saw immediately from their curves that in the region of the zero point the rate of fermentation was so small that it could no longer be measured by the usual gross methodology employed by the aforementioned workers, whereas in the same region the smallest growth rate was always easily measurable. BURK and WOODS saw, in other words, that in the region of the zero pint of their curves the growth test was more sensitive than the usual fermentation test. With refined and adequate methods for measuring fermentation of sugar (glucose) they found, what any physical chemist after a glance at the curve would realize, that even the most slow-growing Morris hepatomas fermented sugar.

The results of DEAN BURK and MARK WOODS were confirmed and extended by other workers with independent methods. PIETRO GULLINO, also in Bethesda, developed a perfusion method whereby a Morris hepatoma growing in the living animal could be perfused for long periods of time, even weeks, by means of a single artery and single vein, and the blood entering and leaving any given tumor could be analyzed. GULLINO found with this method that the slow-growing Morris hepatomas always produced fermentation lactic acid during their growth. This was in contrast to liver, where, as known since the days of CLAUDE BERNARD, lactic acid is not produced but consumed by liver; the difference between liver and Morris tumors in vivo is thus infinite (+ vs. -). GULLINO further found that tumors grow in vivo with diminished oxygen consumption. In summary, GULLINO's findings indicate that the slow-growing Morris hepatomas are partial anaerobes. SILVIO FIALA, a biochemist at the University of Southern California, found that not only did the slow-growing hepatomas produce lactic acid, but also that the number of their oxygen-respiring grana was reduced.
The slow-growing Morris hepatomas are therefore far removed from having refuted the anaerobiosis of tumors. On the contrary, they are the best proof of this distinctive characteristic. For forty years cancer investigators have searched for a cancer that did not ferment. When finally a non-fermenting tumor appeared to have been found in the slow-growing Morris tumors, it was shown to be a methodological error.

Transformation of Embryonic Metabolism into Cancer Metabolism

A third type of experiment, from the institute in Dahlem with coworkers GAWEHN, GEISSLER and LORENZ, is likewise highly pertinent. Having established that anaerobiosis is that property of cancer cells that distinguishes them from all normal body cells, we attacked the question, namely, how normal body cells may become transformed into anaerobes 6)7)8).

If one puts embryonic mouse cells into a suitable culture medium saturated with physiological oxygen pressures, they will grow outside the mouse body, in vitro, and indeed as pure aerobes, with a pure oxygen respiration, without a trace of fermentation. However, if during the growth one provides and oxygen pressure so reduced that the oxygen respiration is partially inhibited, the purely aerobic metabolism of the mouse embryonic cells is quantitatively altered within 48 hours, in the course of two cell divisions, into the metabolism characteristic of fermenting cancer cells. Fig. 2 illustrates the very simple experimental procedure involved.

If one then brings such cells, in which during their growth under reduced oxygen pressure a cancer cell metabolism has been produced, back under the original high oxygen pressure, and allows the cell to grow further, the cancer metabolism remains. The transformation of embryonic cell metabolism into cancer cell metabolism can thus be irreversible, and important result, since the origin of cancer cells from normal body cells is an irreversible process. It is equally important that these body cells whose metabolism has thus been transformed into cancer metabolism now continue to grow in vitro as facultative anaerobes. The duration of our experiments is still too limited to have yielded results of tests of inoculation of such cells back into mice, but according to all previous indications such cells will later grow as anaerobes upon transplantation into animals.

In any case, these experiments belong to the most important experiments in the field of cancer investigation since the discovery of the fermentation of tumors. For cancer metabolism, heretofore, measured so many thousand of times, has now been induced artificially in body cells by the simplest conceivable experimental procedure, and with this artificially induced cancer metabolism the body cells divide and grow as anaerobes in vitro*).

*) The experiments were at once repeated, when they were published, of course without acknowledgment. See for example Th. Goodfriend, D. M. Sokol and N. O. Kaplan, J. molecular Biol. 15, 18, 1966.

In recent months we have further developed our experimental arrangements so that we can measure manometrically the oxygen respiration and fermentation of the growing mouse embryonic cells during the metabolic transformation. Fig. 3 shows the experimental arrangement. We find by such experiments that 35 percent inhibition of oxygen respiration already suffices to bring about such a transformation during cell growth**). Oxygen pressures that inhibit respiration 35 percent can occur at the end of blood capillaries in living animals, so that the possibility arises that cancer may result when too low oxygen pressures occur during cell growth in animal bodies.

**) These experiments show, like the curve of Dean Burk and Mark Woods in Fig. 1, that it is more correct to designate tumor cells as "partial anaerobes" rather than "facultative anaerobes". A body cell is transformed into a tumor cell if only a part of the respiration is replaced by fermentation.

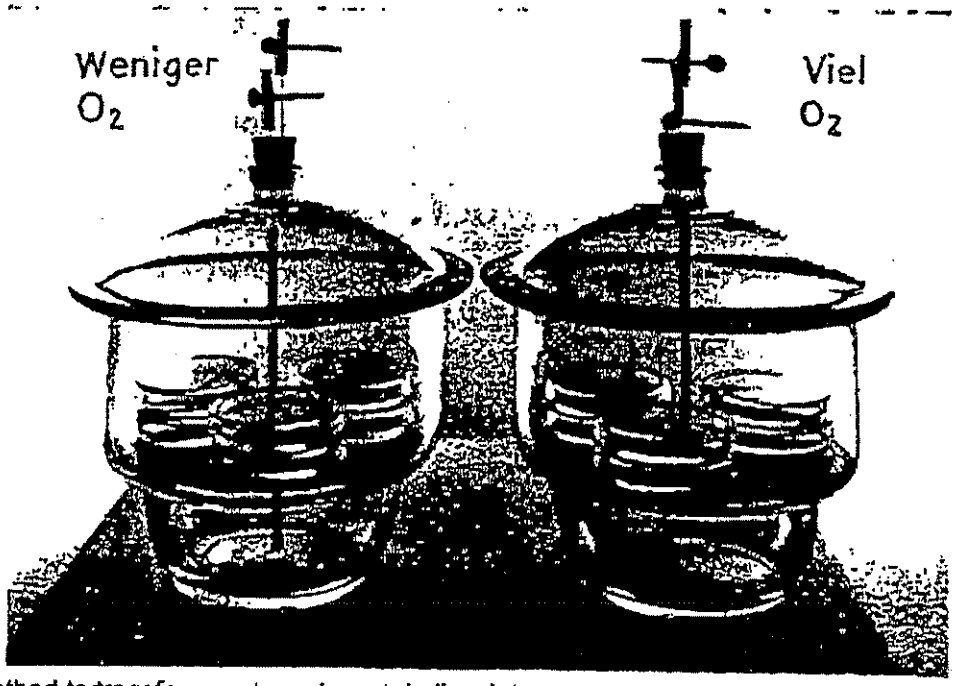


Fig. 2. Method to transform embryonic metabolism into cancer metabolism by decreasing the oxygen pressure

The induction of cancers by solid materials injected into animals is a further experimental indication of this possibility. If one implants discs of solid substances under the skin of rats, the discs will soon be surrounded by capsules of living tissue that will be nourished with blood vessels from the hypodermis. Sarcomas very frequently develop in these capsules. It is immaterial whether the solid discs are chemically plastics, gold, or ivory, etc. What produces the cancer is not the chemical nature of the solid discs, but the special king of blood nourishment supplied to the tissue encapsulating the discs. This blood provision varies with the site and in adequacy within a given animal, and induces cancer from the low oxygen pressure in the encapsulating disc.

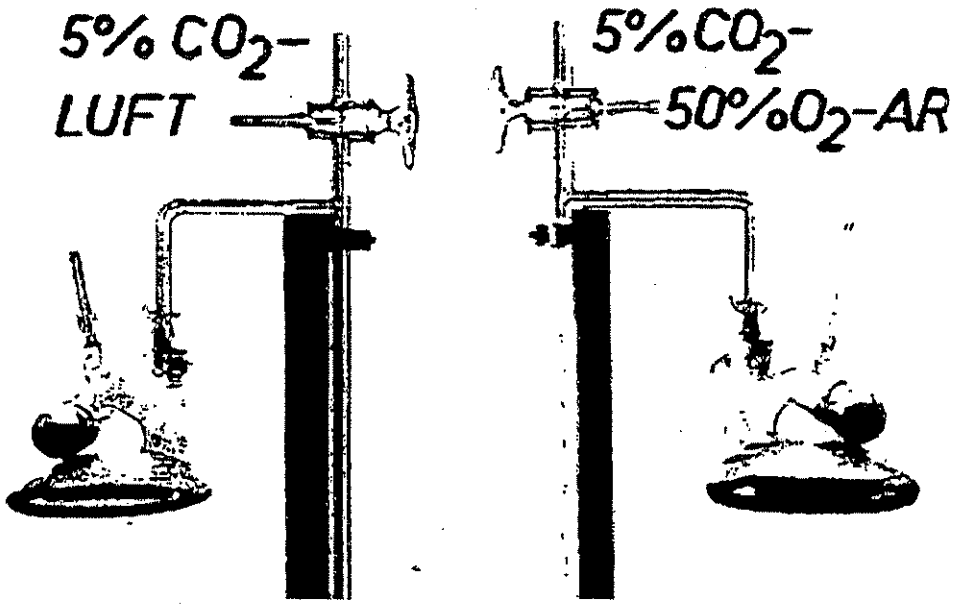


Fig. 3. Method to measure manometrically respiration and fermentation during the transformation of embryonic into cancer metabolism*)

*) The vessels are not shaken, because shaking inhibits growth. Therefore, the oxygen pressure in the liquid phase at the bottom of the vessels is much lower than in the gasphase. For example, when the oxygen pressure in the gas phase was 2000 mm H_2O it was at the bottom of the vessels 130 mm H_2O. (O. Warburg, A. Geissler and S. Lorenz, Zeitschr. Für Naturforschung 20b, 1070, 1965.)

Thermodynamics

If a lowered oxygen pressure during cell growth may cause cancer, or, more generally, if any inhibition of respiration during growth may cause cancer, then a next problem is to show why reduced respiration induces cancer. Since we already know that with a lowering of respiration fermentation results, we can re-express our question: Why does cancer result if oxygen-respiration is replaced by fermentation?

The early history of life on our planet indicates that life existed on earth before the earth's atmosphere contained free oxygen gas. The living cells must therefore have been fermenting cells then, and, as fossils show, they were undifferentiated single cells. Only when free oxygen appeared in the atmosphere - some billion years ago - did the higher development of life set in, to produce the plant and animal kingdoms from the fermenting, undifferentiated single cells. What the philosophers of life have called "Evolution créatrice" has been and is therefore the work of oxygen.

The reverse process, the dedifferentiation of life, takes place today in greatest amount before our eyes in cancer development, which is another expression for dedifferentiation. To be sure, cancer development takes place even in the presence of free oxygen gas in the atmosphere, but this oxygen may not penetrate in sufficient quantity into the growing body cells, or the respiratory apo-enzymes of the growing body cells may not be saturated with the active groups. In any case, during the cancer development the oxygen-respiration always falls, fermentation appears, and the highly differentiated cells are transformed to fermenting anaerobes, which have lost all their body functions and retain only

the now useless property of growth. Thus, when respiration disappears, life does not disappear, but the meaning of life disappears, and what remains are growing machines that destroy the body in which they grow.

But why oxygen differentiates and why lack of oxygen dedifferentiates? Nobody would dispute that the development of plants and animals and man from unicellular anaerobes is the most improbable process of all processes in the world. Thus there is no doubt, that EINSTEIN descended from a unicellular fermenting organism - to illustrate the miracle, molecular O_2 achieved. But according to the thermodynamics of Boltzmann, improbable processes require work to take place.

It requires work to produce temperature differences in a uniformly temperatured gas; whereas the equalization of such temperature differences is a spontaneous process that does not require work. It is the oxygen-respiration that provides in life this work, and dedifferentiation begins at once when respiration is inhibited in any way. In the language of thermodynamics, differentiation represents a forced steady state, whereas dedifferentiation - that is, cancer - is the true equilibrium state. Or, illustrated by a picture: the differentiated body cell is like a ball on an inclined plane, which, would roll down except for the work of oxygen-respiration always preventing this. If oxygen respiration is inhibited, the ball rolls down the plane to the level of dedifferentiation.

But why respiratory energy and not fermentation energy can differentiate, whereas in general, for example in growth, respiratory energy and fermentation energy are equivalent? Obviously, there would be no cancer if there were not this discrimination of fermentation energy, that is, if fermentation like respiration could differentiate. Then, when respiration is replaced by fermentation, fermentation would take over differentiation, and a high state of differentiation would be maintained even in the fermenting body cells.

Chemistry

Physics cannot explain why the two kinds of energy are not equivalent in differentiation; but chemistry may explain it. Biochemists know that both respiration energy and fermentation energy do their work as phosphate energy, but the ways of phosphorylation are different. If one applies this knowledge to carcinogenesis, it seems that only oxidative phosphorylation but not fermentative phosphorylation can differentiate, a result, that may in future explain the mechanism of differentiation.

Yet Biochemistry can explain already today why fermentation arises, when respiration decreases. Figure 4 shows that the pathways of respiration and fermentation are common as far as pyruvic acid. Then the pathways diverge. The endproducts of fermentation is reached by one single reaction, the reduction of pyruvic acid by dihydro-nicotinamide to lactic acid. On the other hand, the endproducts of the oxidation of pyruvic acid, H_2O and CO_2, are only reached after many additional reactions. Therefore, when cells are harmed, it is probable that first respiration is harmed.

In this way the frequency of cancer is explained by reasons of probability.

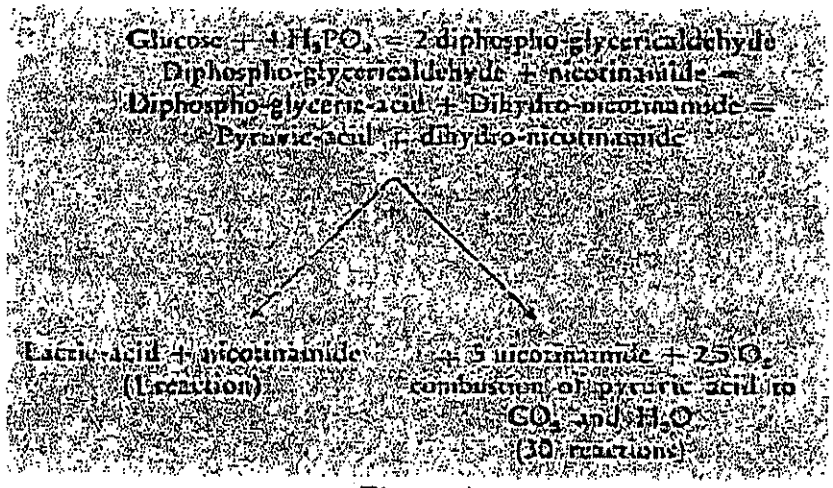


Figure 4

To sum up:

1. Impairment of respiration is frequent than impairment of fermentation because respiration is more complicated than fermentation.
2. The impaired respiration can be easily replaced by fermentation, because both processes have a common catalyst, the nicotinamide.
3. The consequence of the replacement of respiration by fermentation is mostly glycolysis, with death of the cells by lack of energy. Only if the energy of fermentation is equivalent to the lost energy of respiration, is the consequence anaerobiosis. Glycolysis means death by fermentation, anaerobiosis means life by fermentation.
4. Cancer arises, because respiration, but not fermentation, can maintain and create the high differentiation of body cells.

To conclude the discussion on the prime cause of cancer, the virus-theory of cancer may be mentioned. It is the most cherished topic of the philosophers of cancer. If it were true, it would be possible to prevent and cure cancer by the methods of virology; and all carcinogens could be eaten or smoked freely without any danger, if only contact with the cancer virus would be avoided.

It is true that some virus-caused cancer [b]) occur in animals, but no one sure human virus-cancer has been observed so far, whereas innumerable substances cause cancer without viruses in animals and man. Thus viruses do not meet the demands of Pasteur, that is must be possible to trace the prime cause in every case of the disease. Therefore science classifies viruses as remote causes of cancer, leading to anaerobiosis, the prime cause that meets the demands of Pasteur.

b) The chicken Rous sarcoma, which is labeled today as a virus tumor, ferments glucose and lives as a partial anaerobe like all tumors. O. WARBURG, Bioch. Zeitschrift 160, 307, 1925; F. WIND, Klinische Wochenschrift, Nr. 30, 1926.

Many may remember how anaerobiosis as prime cause of cancer was recently disputed emphatically, when one single cancer - the slow Morris hepatomas - was believed (wrongly) to lack in fermentation. In contrast the virus theory is adhered to although all cancers of man are lacking in virus-origin. This means the surrender of the principles of Pasteur and the relapse into bygone times of

medicine.

Applications

Of what use is it to know the prime cause of cancer? Here is an example. In Scandinavian countries there occurs a cancer of throat and esophagus whose precursor is the so-called Plummer-Vinson syndrome. This syndrome can be healed when one adds to the diet the active groups of respiratory enzymes, for example: iron salts, riboflavin, nicotinamide, and pantothenic acid. When one can heal the precursor of a cancer, one can prevent this cancer. According to ERNEST WYNDER 3) of the Sloan-Kettering Institute for Cancer Research in New York, the time has come when one can exterminate this kind of cancer with the help of the active groups of the respiratory enzymes.

It is of interest in this connection that with the help of one of these active groups of the respiratory enzymes, namely nicotinamide, tuberculosis can be healed quite as well as with streptomycin, but without the side effects of the latter [c]). Since the sulfonamides and antibiotics, this discovery made in 1945 is the most important event in the field of chemotherapy generally, and encourages, in association with the experiences in Scandinavia, efforts to prevent cancer by dietary addition of large amounts of the active groups of the respiratory enzymes. Since there can scarcely be overdosage, such experiments can do no harm.

c) V. CHORINE: C. R. sci. Paris, 220, 150 (1945). – H. FUST and A. STUDER, Schweizerische Z. für allgemeine Pathologie, Band 14; Fasc 5 (1951).

I would like to go further and propose always making dietary additions of large amounts of the active groups of the respiratory enzymes after successful operations when there is danger from metastatic growths. One could indeed never succeed in redifferentiating the dedifferentiated cancer cells, since during the short duration of human life the probability of such a back-differentiation is zero. But one might increase the respiration of growing metastases, and thereby inhibit their fermentation, and - on the basis of the curve of DEAN BURK and MARK WOODS obtained with the Morris hepatomas - thereby inhibit the growth of metastases to such an extent that they might become as harmless as the so-called "sleeping" cancer cells in the prostates of elderly men.

A Second Example of Application

The physicist MANFRED VON ARDENNE has recently attacked the problem of the therapy of cancer. ARDENNE discovered that cancer cells owing to their fermentation, are more acid – inside and on their surface – than normal cells and hence are more sensitive to high temperatures. On this basis, he and his medical colleagues have treated cancer patients, after surgical removal of the primary tumors, by raising the body temperature of the patients to about 109° Fahrenheit for an hour, in the hope that the metastases will then be killed or their growth so slowed up as to become harmless. It is not yet decided whether this idea can be described as a practical success. But the provisional work of ARDENNE is already of great significance in a field where hopes of conventional chemotherapy have been dimmed but might be brightened by combination with extreme or moderate hyperthermy.

A third application. According to an estimate by K. H. Bauer of the Cancer Institute in Heidelberg, at least one million of the now living twenty five million male inhabitants of West Germany will die of cancer of the respiratory tract; still more will die from other cancer. When one considers that cancer is a permanent menace, one realizes that cancer has become one of the most dangerous menaces in the history of medicine.

Many experts agree that one could prevent about 80% of all cancers in man, if one could keep away the known carcinogens from the normal body cells. This prevention of cancer might involve no expenses, and especially would require little further research to bring about cancer prevention in up to 80 percent *).

*) Since this estimate was published, some though 80% even to low. Yet prevention remained taboo and early diagnosis was the only consolation that was offered.

Why then does it happen that in spite of all this so little is done towards the prevention of cancer? The answer has always been that one does not know what cancer or the prime cause of cancer be, and that one cannot prevent something that is not known.

But nobody today can say that one does not know what cancer and its prime cause be. On the contrary, there is no disease whose prime cause is better known, so that today ignorance is no longer an excuse that one cannot do more about prevention. That prevention of cancer will come there is no doubt, for man wishes to survive. But how long prevention will be avoided depends on how long the prophets of agnosticism will succeed in inhibiting the application of scientific knowledge in the cancer field. In the meantime, millions of men must die of cancer unnecessarily.

Literature to Preface of Second Edition:

1. WILLSTAETTER, WIELAND and EULER, Lectures on enzymes at the centenary of the Gesellschaft Deutscher Naturforscher. Berichte der Deutschen Chemischen Gesellschaft, 55, 3583, 1922. The 3 lectures of the 3 chemists show that in the year 1922 the action of all enzymes was still a mystery. No active group of any enzyme was known.
2. OTTO WARBURG, Biochem. Zeitschrift, 152, 479, 1924.
3. OTTO WARBURG, Heavy Metals as prosthetic groups of enzymes, Clarendon Press, Oxford, 1949.
4. OTTO WARBURG, Wasserstoffübertragende Fermente, Verlag Werner Sänger, Berlin, 1948.
5. DEAN BURK, 1941. On the specificity of glycolysis in malignant liver tumors as compared with homologous adult or growing liver tissues. In Symposium of Respiratory Enzymes, Univ. of Wisconsin Press. pp. 235-245,1942. DEAN BURK, Science 123,314,1956. Woods, M. W., Sandford, K. K., Burk, D., and Earle, W. R. J. National Cancer Institute 23, 1079-1088, 1959. DEAN BURK, Burk, D., Woods, M. and Hunter, J. On the Significance of Glucolysis for Cancer Growth, with Special Reference to Morris Rat Hepatomas. Journ. National Cancer Institute 38, 839-863, 1967.
6. O. WARBURG und F. KUBOWITZ, Bioch. Z. 189, 242, 1927; H. GOLDBLATT und G. CAMERON, J. Exper. Med. 97, 525, 1953.
7. O. WARBURG, 17. Mosbacher Kolloquium, April 1966. Verlag Springer, Heidelberg, 1966.
8. O. WARBURG, K. GAWEHN, A. W. GEISSLER, D. KAYSER and S. LORENZ, Klinische Wochenschrift 43, 289, 1965.
9. O. WARBURG, Oxygen, The Creator of Differentiation, Biochemical Energetics, Academic Press, New York, 1966.
10. O. WARBURG, New Methods of Cell Physiology, Georg Thieme, Stuttgart, and Interscience Publishers, New York, 1962.

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Cancer Report

Cancer does not develop for some unknown reason. Once you understand why and how it develops, and learn how to change the internal environment in your body so that it it can become a place where health flourishes, not cancer, it may never be too late to get healthy again. Cancerous cells are always developing. They always have. The immune system evolved and developed components whose job it is to seek out and destroy cancerous cells. They have been around as long as mankind. Just that there is a lot more of it now. Cancer is a perfectly natural occurence.

Cancer tumors begin when more cancerous cells are being created than an overworked, depleted immune system can destroy. Constant exposure to tens of thousands of manmade chemicals from birth onward, along with living conditions that do not create a healthy internal environment, leads to the creation of excessive numbers of cancerous cells. Combine this increase with an immune system weakened by our modern diet of refined and overprocessed food, mineral depleted soils, overworked and depleted by high stress living conditions. What you get is a malfunctioning immune system that is not capable of destroying the excessive numbers of cancerous cells that develop. Some, sooner or later, survive and multiply. And you have cancer.

So there is really no such thing as a cure for a cancer. As it is a natural process. Just like there will never be a cure for digestive upset if you continually overeat, or from feeling lousy if you drink too much alcohol. What needs to be done is to interrupt this process so that your body becomes more healthy and moves away from a cancer friendly internal environment. The more health you need, the more you may need to to do. So...

What you will be reading in this report isn't about a cure for cancer. It isn't medical advice. You can still do whatever your doctor may be telling you to do. Consider this information as something additional, outside the realm of medicine, that may improve your chances of recovery. Just like exercise improving your chances of recovery from heart disease.

It's about how to make your body more healthy so that it is more capable of fighting cancer. Sort of like saying if you eat more broccoli, or more servings of fresh fruits and vegetable, you'll give your body more nutrients that will possibly make you healthier, and thus, naturally better able to deal with or prevent cancer. More specifically, this report is about what you can do to create healthy internal

environment in your body.

Change your body's internal environment to one where cancer cannot thrive, and cancer may have a much harder time surviving. Interestingly enough, there are just a few fundamentals that can make a big difference in that internal environment, turning it towards health. Revolving around the alkaline/acidity levels and oxygen levels in the cells.

Just as we couldn't live on Mars with no oxygen, research shows that cancer can't exist in cells when there *is enough* oxygen or when the pH is where it is supposed to be. Not overly acidic. Unfortunately, all too often our cells are low in oxygen and acidic. Fortunately it may not be that difficult to adjust the internal environment so that cancer can't easily thrive or survive. Doing this in conjunction with strengthening the immune system may strengthen the body and weaken the cancer to the point where the cancer no longer is multiplying faster than the immune system can destroy it, and eventually one regains health.

There are lots of stories of people naturally recovering from cancer. In all sorts of different ways. There are also lots of people who try many different avenues in dealing with their cancer and still die. The trick is to find what is the *most* effective support for the body. That deals with the fundamental issue of creating the right type of internal environment. That supports health most strongly so that health begins to thrive.

Much as I enjoy reading or hearing testimonies, you will read none in this report. The government now considers them product claims. Rather, I will, in this report focus on making as clear as I can, some of the underlying fundamental causes leading to most any cancer.

Carcinogens, toxins and other cancer causing agents, may actually be, in a sense, secondary causes of cancer. An underlying cause of all cancers may have been discovered by the renowned scientist Otto Warburg over 50 years ago. He actually won two Nobel Prizes for discovering the clues to why cancer develops. His continued research, and the research of a few other cancer investigators, back up his contention. Cancer development seems to be connected to...

Cellular Oxygen Levels

Lack of cellular oxygenation may be a prime factor leading to cancer.

In 1931 Dr. Warburg won his first Nobel Prize for work proving cancer is caused by a lack of oxygen respiration in cells. He stated in an article titled *The Prime Cause and Prevention of Cancer* that "the cause of cancer is no longer a mystery, we know it occurs whenever any cell is denied 60% of its oxygen requirements."

"Cancer, above all other diseases, has countless secondary causes. But, even for cancer, there is only one prime cause. Summarized in a few words, the prime cause of cancer is the replacement of the respiration of oxygen in normal body cells by a fermentation of sugar. All normal body cells meet their energy needs by respiration of oxygen, whereas cancer cells meet their energy needs in great part by fermentation. All normal body cells are thus obligate aerobes, whereas all cancer cells are partial anaerobes."

Some causes of poor oxygenation include a buildup of carcinogens and other toxins within and around cells, which blocks and then damages the cellular oxygen respiration mechanism. Clumping up of red blood cells slows down the bloodstream and restricts flow into capillaries. Which also causes poor oxygenation. Even lack of the proper building blocks for cell walls, essential fatty acids, restricts oxygen exchange.

What Warburg and other scientists found in their research was that the respiratory enzymes in cells, used to make energy aerobically, die when cellular oxygen levels drop. When this happens, the cell can no longer produce energy aerobically. So, if the cell is to live, it must, at least partially, ferment sugars, producing energy anaerobically. According to Warburg, cells that produce energy by fermenting sugars may turn cancerous. Warburg's contention is this...

Cells must switch to producing energy anaerobically because low oxygen levels have lead to the death of the respiratory enzymes needed to produce energy aerobically. When this happens, the cells cannot produce enough energy to maintain their ability to function properly. So they lose their ability to do whatever they need to do in the body. Fermentation allows these cells to survive, but they can no longer perform any functions in the body or communicate effectively with the body. Consequently, these cells only multiply and grow. And may become cancerous. Or perhaps it would be more accurate to say, they evolve into cancer cells.

I went online to research what Dr. Warburg was actually saying. It seemed like most every oxygen site was quoting a sentence of two from him, but I thought I should find out what he was really saying. And learned the above information.

It turns out that decades ago, two researchers at the National Cancer Institute, Dean Burn and Mark Woods, (Dean actually translated some of Warburg's speeches.) conducted a series of experiments where they measured the fermentation rate of cancers that grew at different speeds. What they found supported Dr. Warburg's theory. The cancers with the highest growth rates had the highest fermentation rates. The slower a cancer grew, the less it used fermentation to produce energy.

[Thinking about this, there could well be an additional cause in turning a cell cancerous. Something that flips a cell fermenting energy anaerobically into a cancer cell. One that we don't know about. However, lack of oxygenation, be the underlying condition that must be present for the development of a cancer cell, is the primary issue. Get enough oxygen into cells so that they don't have to produce

energy through fermentation, and the cell won't turn into a cancer cell.]

Naturally Warburg's contention was challenged and tested by other scientists.

There were some researchers who claimed that this theory was not valid because they had measured a particularly slow growing cancer, and found no fermentation at all. And if cancer could grow with no fermentation, then fermentation, or lack of oxygen respiration, was not the cause of cancer. Dean Burn and Mark Woods checked those results. Using more sophisticated equipment, they determined that the test these researchers used to measure fermentation levels was not accurate enough to detect fermentation at low levels. Their testing, using newer and more accurate equipment, showed that even in those very slow growing cancer cells, fermentation was still taking place, at very low levels.

This was also confirmed by Pietro Gullino, also at the National Cancer Institute, who devised a test which showed that this slow growing cancer *always* produced fermentation lactic acid. Silvio Fiala, a biochemist from the University of Southern California, also confirmed that this slow growing cancer produced lactic acid, and that it's oxygen respiration was reduced.

This theory makes sense. It also explains why there is *never* cancer of the heart. There are brain cancers, bladder cancers, cancers of every organ in the body. But have you ever heard of heart cancer? There wouldn't be, not it this theory is correct.

That's because the body will not allow heart cells to produce energy anaerobically. Here's why. When you are working, or working out, and your muscles start to ache with pain, they have switched to temporarily producing energy anaerobically as they can't get enough oxygen. This creates a lactic acid buildup which eventually shuts down the muscle by causing pain.

If this were to take place in heart when it was working hard, lactic acid would build up in the heart. Which would shut down the heart, as if you were having a heart attack, and you die. To prevent this, the body doesn't allow heart muscles to produce energy through anaerobic fermentation. Heart cells just stop working when they can't produce energy using oxygen. Consequently, they don't get cancer because their cells cannot product energy through fermentation. Which means that they can't lose their functionality and grow in an uncontrolled manner, turning into cancer. In a sense, they do lose functionality when they stop working, but in this case they can't do anything, so they can't turn into cancer cells.

Further research into Warburg's theory showed that when oxygen levels were turned down, cells began to produce energy anaerobically, and ultimately became cancerous when levels went low enough. It took a reduction of 35% in oxygen levels for this to happen.

J.B. Kizer, a biochemist and physicist at Gungnir Research in Portsmith, Ohio explains, "Since Warburg's discovery, this difference in respiration has remained the most fundamental (and some say, only) physiological difference consistently found between normal and cancer cells. Using cell culture studies, I decided to examine the differential responses of normal and cancer cells to changes in the oxygen environment.

"The results that I found were rather remarkable. I found that... "high 02 tensions were lethal to cancer tissue, 95 percent being very toxic, whereas in general, normal tissues were not harmed by high oxygen tensions. Indeed, some normal tissues were found to require high 02 tensions. It does seem to demonstrate the possibility that if the 02 tensions in cancer tissues can be elevated, then the cancer tissue may be able to be killed selectively, as it seems that the cancer cells are incapable of handling the 02 in a high 02 environment."

I bring up this research because there are scientists and others who deny that Warburg's theory is valid because of the original inaccurate measurement of the amount of fermentation in slow growing cancer. In fact, I went to Quackwatch and read a scientist claiming there was no research at all supporting Warburg's theory. So much for his research.

Research Implications

Probably the main implication of this research is that an effective way to support the body's fight against cancer may be to get as much oxygen as you can into the cells. At the very least increase, getting more oxygen into cells, could possibly help slow down the growth of cancer cells. Which could give the body more time to kill them off. Raising the oxygen levels of the still normal cells might also help prevent them from becoming cancerous. And according to one of those research studies, increasing oxygen levels in cancer cells may possibly help kill those cells.

A nurse who works in medical research said, "*It's so simple. I don't know why I never thought of it before. When we're working with cell cultures in the lab, if we want the cells to mutate, we turn down the oxygen. to stop them, we turn the oxygen back up.*"

Ma Lan, MD and Joel Wallach DVD, point out that one type of white blood cells kills cancer cells by injecting oxygen creating hydrogen peroxide into the cells.

There is a bit more to this oxygenation story.

If you remember, according to Warburg, it is increased amounts of carcinogens, toxicity and pollution that causes cells to become so toxic they don't uptake oxygen very well, and leads to this breakdown of cellular respiration. So let's take a closer look at that.

Acidity and Alkalinity

Again, there seems to be plenty of research showing that cancer thrives in an acidic environment, and doesn't do well in a more alkaline environment. Cancer cells produce lactic acid as a by-product of fermentation, which makes them highly acidic. Thereby, taking action to make the body more alkaline may be of benefit in dealing effectively with cancer. Unfortunately...

The majority of the foods and drinks we consume are acidic, with colas and other soft drinks being highly acidic. So unless you are eating a special diet, your body is *way* too acidic. And is a very good environment for cancer to thrive in.

According to Keiichi Morishita in his book, Hidden Truth of Cancer, If blood starts to become acidic, then the body deposits the excess acidic substances in cells so that the blood will be able to maintain a slightly alkaline condition. This causes those cells to become more acidic, and also causes a decrease in their oxygen levels.

Over time, he theorizes, these cells increase in acidity and some die. These dead cells themselves turn into acids. However, some of these acidified cells may adapt in that environment. In other words, instead of dying - as normal cells do in an acid environment - some cells survive by becoming abnormal cells. These abnormal cells are called malignant cells. Malignant cells do not correspond with brain function nor with our own DNS memory code. Therefore, malignant cells grow indefinitely and without order. This is cancer.

As you can see, what he seems to be describing, though from a different point of view, is the process by which low oxygen levels turns some cells cancerous. It is interesting to note the connections here. Alkaline water (including the water in cells) can hold a lot of oxygen. Acidic water (or cells) can hold very little oxygen. So the more acidic your cells are, the less oxygenated they will be.

Sang Whang, in his book *Reverse Aging*, points out that toxins are acidic. If the blood is already too acidic, the body must take the toxins out of the blood and deposit them in cells, to keep the blood the right pH. And it cannot release toxins into the blood to detoxify the cells, when the blood is too acidic. All of which helps to cause acidic, poorly oxygenated cells which may, at some point, from excess acidity and lack of oxygen, turn cancerous.

By the way, it is not so easy to get oxygen into cells. Most approaches don't work well. Breathing oxygen is still limited by the amount of hemoglobin and acidity for getting oxygen into the cells. And Dr. Whittaker points out, quite rightly, that liquid oxygen supplements also don't work because they don't get oxygen into the cells. He explains that a delivery mechanism is needed to get the oxygen into the cells. And that getting oxygen into the stomach or the blood won't get it into the cells. I agree with him entirely.

The Immune System And Cancer

The other aspect of dealing with cancer is supporting the immune system. After all, for most of your life the immune system has successfully dealt with cancer cells. Usually it becomes worn out and ineffective and unable to deal with the cancer cells before cancer takes hold and thrives.

So it may be quite important to strengthen the immune system so that it can better fight cancer. Especially if one is taking medical treatments that further wipe out the immune system, and make the body more acidic to boot. Current cancer treatments seem to make no sense once you understand the basics of cancer.

Some natural supplements are better than others at supporting the immune system and creating health in the body. Any can help support the body's fight against cancer, however the trick is the use the ones that may help the most, or help the most people. And the other concern, if you have cancer, is to take make sure you take enough of them. Generally 5 to 10 times the normal supplemental amounts seem to work best for food based supplements.

Cancer vs. Health

A natural approach to cancer is based on making the body healthier. It works on the basic conditions that cause cancer to develop. Strengthening a depleted, worn out, under energized immune system that is not capable of killing cancer as fast as they are multiplying. And changing the internal environment of the body so that the cancer cells have a harder time surviving because the conditions that allow them to grow so prolifically have changed.

Cancer is not something to play around with. The more you have to overcome, the more you have to do. The key to getting rid of cancer, any cancer, may be to move your body's internal environment towards health, and away from an environment conducive to the growth of cancer.

It may never be too late to strengthen your body to fight cancer. The more cancer you have, the more nutrients and energy you'll need, to move the body back to a state of health. The greater your action will have to be.

Again this isn't a treatment for a particular cancer. It is about how to make yourself healthier as quickly as possible so that your body can take care of whatever ill health condition needs to be taken care of.

And if you have any questions about the information in this report, feel free to call or email me.

Warm regards,

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Disclaimer: These statements have not been evaluated by the Food and Drug Administration. The products and information contained herein are not intended to diagnose, treat, cure, or prevent any diseases or, medical problems. It is not intended to replace your doctor's recommendations. The information is provided for educational purposes only. Nutritional benefits may vary from one person to another.

The Osteoporosis Education Project has conduct several research projects on acid alkaline balance. One project was collaborative research with Dr. Susan Whiting of the University of Saskatchewan on the relationship between first morning urine pH measurement and net acid load. For an abstract of these research findings as presented at the 2002 ASBMR meetings, see the First Morning pH abstract below. Investigators Susan Whiting, Ph.D., Janet Bell, and Susan E. Brown, Ph.D., CCN.

First Morning Urine Measured With pH Paper Strips Reflects Acid Excretion

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Net acid excretion (NAE) is implicated in bone loss, as increased calcium loss is seen with a high net acid excretion. Dietary protein is identified as a significant producer of acid whereas fruit and vegetable may counteract this effect through the production of metabolizable organic anions which buffer acid. Determination of NAE is important in recognizing the effect diet may have on bone. Most commonly, a 24-hour urine collection is obtained for measurement of NAE where NAE is measured as titratable acidity minus bicarbonate (TA-bicarb) plus ammonium ($NH4+$). However, this measurement can be inconvenient and pH measured on first morning urine with semi-quantitative paper strips may be a practical estimator of NAE. We recruited 23 (4M, 19F) healthy subjects age 20-50 y who recorded dietary intake for a day during which they collected urine from approximately 7 am to 11 pm in one container ("day") and approximately 11 pm to 7 am ("overnight",ON) in a separate container. The first morning void contained ON urine. Subjects also provided a two-hour fasting urine at 9 am. pH paper strips (colorpHast ®, EM-Reagents, range 4-7) were used to measure pH of the ON urine, as would be done in practice. A second set of strips (pH range 6.5-10) was used if initial pH read high. Although measurement with pH paper strips was not significantly correlated with 24-hr NAE, there was a significant correlation with 24-hour TA-bicarb ($r= -0.466$, $p<0.025$). Further, pH strip measures were significantly correlated with ON NAE ($r= -0.710$, $p<0.005$). We noted that ON NAE was correlated with total NAE ($r=0.504$, $p<0.014$). We conclude there is useful information is measuring first morning urine pH (which provides pH of urine formed overnight) to obtain an estimate of acid excretion. pH paper strips appear to be useful in the absence of longer (more invasive) urine collections.

Acid-Alkaline Balance and Its Effect on Bone Health

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In our bones, most North Americans are in woeful shape. Our bone health crisis worsens each year, despite intensive public health and disease treatment efforts. In this article, we will suggest that the preoccupation with the consequences of osteoporosis, rather than a focus on its fundamental causes, underlies our inability to solve the contemporary epidemic of poor bone health.

Osteoporosis can be seen as a "bidden tax of high-tech living." We pay this tax as a consequence of chronic metabolic acidosis, which robs us of our mineral reserves and impairs efforts to rebuild the bone matrix. The basis of, support for, and a comprehensive response to this situation are detailed below.

Following are the basics of bone growth and turnover. Understanding these basics can helps prevent or repair osteopenia and osteoporosis, as detailed later in this article.

Bone growth largely halts after puberty. However, "bone tissue in adults is not dormant -- our bones are continuously being remodeled through repeated cycles of destruction and rebuilding."[1] Healthy people balance osteoclastic bone destruction with osteoblastic bone rebuilding.[2] The normative remodeling half-life of bone is five years. This means that every 10 years a healthy person will have an entirely new skeletal structure.

While bone-remodeling processes constantly attempt to renew bone, the skeletal structure of a typical North American senior citizen is not healthy. In the United States, half of all Caucasian women aged 65 and over, and at least one in five men, will experience one or more osteoporotic fractures during their lifetime.[4] Currently, about 10,000,000 North Americans have been diagnosed with osteoporosis. An additional 18,000,000 North Americans have osteopenia and face a high risk of bone fracture or related complications. Direct medical costs related to osteoporosis exceed $14 billion per year. This represents about one-third of the total cost to our nation for osteoporosis-related ill health.

Furthermore, in 1999, 1,167 scientific articles on bone health and osteoporosis were added to the index Medicus database, representing about half of the peer-reviewed studies published on the subject that year. According to the National Institutes of Health (NIH) Public Information Office, $136.7 million federal dollars were spent in 1999 on osteoporosis research. Despite these great efforts, a solution to our current bone health crisis still evades us, and fracture incidence increases, particularly in younger people.[3]

The Link Between Osteoporosis and Metabolic Acidosis

From a broad systemic perspective, primary osteoporosis is seen largely as secondary to metabolic acidosis. First, we will discuss the causal link between acid-alkaline balance and bone health, and then we will present options for restoration and maintenance of bone health throughout the full life span.

In contemporary Western society, diet/lifestyle-induced metabolic acidosis is more the rule than the exception. As detailed below, the excess acid load promoting metabolic acidosis is acquired by:
1. Dietary choices (excess protein, fat, phosphate/phosphoric acid, and sulfate/sulfuric acid);
2. Maladaptation to stress (distress-induced excess cortisol and adrenaline); and
3. Immune hypersensitivity (delayed allergy) reactions.

For 80 years, it has been repeatedly confirmed that bone responds to an acid load by dissolving its basic buffering mineral salts. For background, the average adult skeleton contains a large but finite amount of Ca^{2+} (50-65,000 mEq, 99% of total body stores) and Mg^{2+} (1,060-1,600 mEq, 50% to 80% of body stores). Bone minerals serve as a sizeable reservoir of buffer, usable in the control of plasma pH. Extensive research has documented the following[8-18]:

1. Urinary calcium excretion is associated with bone loss.
2. Urinary calcium loss in the face of an acid load strongly suggests cellular potassium and sodium deficits.
3. Bone loss is accelerated in the face of magnesium deficit.
4. Urinary calcium excretion parallels total acid excretion until substantial calcium and magnesium deficits accumulate.
5. Upon significant depletion of buffering mineral salts, compensation for acid load is reduced, intracellular and first-morning urine pH is concomitantly reduced, and the consequences of metabolic acidosis are accelerated. As an equilibrated specimen, first-morning urine pH is a useful clinical approximation of the cellular and systemic acid/alkaline state.

Less appreciated, however, are the following facts:

1. A variety of alkaline buffering salts (including those of sodium, potassium, zinc, and other minerals) are stored in bone. They are also lost from bone in the obligatory buffering of excessive metabolic acids.[19]
2. The contribution of contemporary dietary patterns to the induction of excess metabolic acids has been clinically underestimated. These fixed acids, which must be neutralized with alkaline buffering mineral salts, are largely the result of less healthy dietary choices.[20,21]

3. The mineral deficits in our soil and water reduce the availability of minerals in the conventional food supply.[22,23]
4. Compensated chronic metabolic acidosis is more the rule than the exception. The results are depletion of bone tissue and a disposition to chronic illnesses.[24-28]
Thus, although osteoporosis is a complex and often multi-faceted disorder, we propose that primary osteoporosis is largely secondary to acquired and reversible chronic metabolic acidosis.

Small Change, Big Impact

Bone is sensitive to small changes in pH. In *vitro* studies document that even one-tenth of a point drop in pH does the following.[64,65]

1. Greatly stimulates osteoclastic activity
2. Inhibits osteoblastic action; and
3. Induces a multifold bone mineral loss.

A 500%-900% increase in osteoclast cell-meditated rat bone resorption was noted with just a 0.2 pH unit change.[66] Acidosis also induces mineral dissolution, independent of osteoclastic activity. For example, a human study on acute fasting showed a venous pH decrease from 7.37 to 7.33 (4/100th of a pH unit). This caused a significant calcium release from bone, which was independent of osteoclast of PTH activity.[67]

Acid-Alkaline Bone and Body Balance

It has been said that "the body is alkaline by design, but acidic by function."[29] The human body has also been described as largely dilute seawater encapsulated in a membrane skin. Our immune defense and repair mechanisms, and a host of cell and system enzyme catalysts, all do their best in an exquisitely narrow pH range. The healthy pH range of oxygenated arterial blood is 7.35 to 7.45, and that of the carbon dioxide-laden venous blood is 7.31 to 7.41. To remain viable, the body must remain slightly alkaline. The viable human arterial blood pH range is just 7.4 ± 0.5 pH units. Even minor variations from these values are biologically costly.

For intracellular cytoplasmic pH, the healthy range is 7.4 ± 0.1. An acidic tilt to cellular pH alters cellular metabolism dramatically and adversely. This results in:

- Swelling and impaired function of mitochondrial electron transport, with reduced ATP energy production and more rapid ATP energy consumption;[30]
- Increase in intracellular free water with less efficient metabolism, protein synthesis, and increased membrane free radical production;[31]
- Increase in interstitial "third space" water (fluid retention), particularly in any susceptible (distressed) organ;[32]
- Accelerated bone resorption;[33]Reduced bone formation;[34]
- Nitrogen wasting (accelerated catabolism);" and
- Suppression of growth hormone and other pituitary hormones.[36]

Although alkaline by design, everyday metabolic processes produce some 70,000 mmol of protons (H+) daily. For the most part, these H+ do not accumulate in the body because of the body's elegant buffering systems, and because acids are generally formed with a partner that aids in their removal. In fact, while an enormous number of H+ are produced daily, most of them are balanced by bicarbonate production. The amount of free H+ is tiny, yet significant, in terms of health maintenance and disease risk.

In most individuals, the source of net acid load is from the metabolism of protein (when its consumption exceeds 60g/day) and long-chain fatty acids (when they comprise more than 20 % of calories in the diet). A marker of net acid production is the extent of degeneration of sulfur-containing amino aids: cysteine, cystine, and methionine. More accurately, any of the seven acidic amino acids (aspartate, glutamate, cysteine, cystine, proline/hydroxyproline, serine, and threonine), plus the keto-acids produced from amino acid metabolism, contribute to the body's fixed, organic acid load. [37] The metabolism of these amino acids produces H+ without buffering partners. These H+ accumulate and must be neutralized by matching buffering elements from the body. The buffering elements include the organize anions (usually as K+ or other mineral salts) in fruits, vegetables, lentils/pulses, herbs, and spices. These include metabolically alkaline-forming citrate, malate, succinate, and fumarate.[38] In addition, short- and medium-chain fatty acids reduce net acid burden by "soaking up" acetate and 2-carbon acidic units in the cells.

The Role of Bone In Systemic Acid-Alkaline Balance

It is well-known that the skeleton contains 99% of the body's calcium. However, bone also contains substantial amounts of sodium, potassium, magnesium, citrate, and carbonate. This means that the bone of a typical, healthy, 70 kg (154 lbs)adult contains.[39-44]

1. 1,065-1,400 mmol of sodium = 1,065-1,400 meq sodium (37%-49% of the body's sodium)
2. 22-62 mmol of potassium = 22-62 meq potassium (0.1%-0.2% of the body's potassium)
3. 530-800 mmol of magnesium = 1,060-1,600 meq magnesium (53%-80% of the body's magnesium)
4. 3,500-5,000 mmol of carbonate = 7,000 to 10,000 meq carbonate (59%-83% of the body's carbonate)

Half of these are located on the bone crystalloid surface and in the hydration shell of bone. These buffering minerals are available for rapid exchange with the general extracellular fluid (ECF). The ECF of bone also contains a potassium concentration 25 times that of general ECF, and thus is a major source from which the body can draw potassium. This potassium is neither incorporated into the bone mineral phase, nor bound to collagen. Therefore, it is completely exchangeable with the potassium of systemic ECF. Potassium accumulates both in the bone ECF and in the bone hydration shell and, overall, is about 60% available for immediate systemic mobilization. [45,46] Thus, a wide range of buffering substances are stored in and around the bone. These are

available to neutralize excess acid products, unless (or until) they become depleted through lack of "alkaline way" replenishment.

Initially, the acid load involves significant changes in the bone content of carbonate, sodium, and potassium, but not calcium. In early phase (fully compensated) acidosis, protons exchange with sodium and potassium, providing a "first line" of buffering defense. Chronic overproduction of acid (chronic metabolic acidosis) leads to depletion of the sodium and potassium buffers. When this occurs, calcium and magnesium cations, along with carbonate, become the major source of buffers.[47] This means that when we see accelerated calcium and magnesium loss, there has been a prolonged period of excess acid production and depletion of critical sodium and potassium reserves.

Sources of Acidic Load

The major recognized sources of net acid load in the body are:

1) Diet
   a) Protein consumption above 60g/day
   b) Dietary phosphate/phosphoric acid
   c) Dietary sulfate
   d) Long-chain fatty acids in excess of 15%-20% of total dietary calories
2) Distress (excess cortisol and adrenaline)
3) Delayed immune system reactions (from delayed immune sensitivities/reactions)

Net Acid Excess (NAE) from the North American Diet

Our contemporary diet commonly produces an excess load of fixed acids of 100 to 200 mEq per day.[48-50] For example, analysis done by Remer and Manz found that a diet containing 120 grams of protein yielded a net acid excretion of 135.5 mEq/day. Two "moderate" protein diets (95g/day protein) yielded an NAE of 69 to 112 mEq/day. A lactovegetarian "low" protein diet (49g/day protein) yielded an NAE of 24 mEq/day. Thus, dietary choice influences net acid production. High-protein diets produce a six-fold (600%) increase in NAE. This results in low first-morning urine pH, indicating that buffering functional reserve is deficient, and that the risk of metabolic acidosis is correspondingly increased.[51]

For example, as Barzel and Massey[52] calculate, the pH of colas with phosphoric acid is 2.8 to 3.2. However, the kidney cannot excrete urine with a pH much lower than 5, without significantly damaging the genitourinary tract. To achieve a urinary pH of 5, a 12 oz. (330mL) can of cola would have to be diluted 100-fold, requiring an additional 33 liters of urine. Otherwise, a corresponding amount of buffer must be drawn from the body to neutralize the excess acid.

The body routinely buffers the acidic beverage with sodium and potassium if reserves permit, then with a corresponding loss of calcium, magnesium, and other minerals, as available. The buffering needed for one can of cola is the same amount of buffering capacity found in 4 Tums™ tablets. Fruit "spritzers" and naturally carbonated mineral waters, by contrast, do not add this acid burden to the body.

Finally, in addition to the recognized acid-producing precursors, we extend the metabolic balance equation to include the additional acid produced by excess immune (delayed hypersensitivity) reactions, and the effects of distress (excess cortisol and adrenaline). In some individuals, these also add significantly to the total net acid production-excretion. Since calcium is activating (sympathomimetic), supplementation primarily with calcium is clinically both unwise and unproductive. It could accelerate acid production and buffering mineral loss.

Net Acid Excess (NAE) The Body Can Buffer

For their excretion, NAE must be buffered with alkaline agents derived from the diet. Thus, our NAE buffering capacity is diet-dependent. Classic studies show that the body can neutralize about 50 mEq of these fixed metabolic acids per day from an assumed "ideal" North American intake of fruits and vegetables.[53] When fruit and vegetable consumption is reduced, less than the 50 mEq of fixed acids can be buffered without going into tissue alkaline reserves. Equally, when protein intake is more than 60g/day, more acid is produced. Today, our daily NAE is commonly two to four times higher than this standard 50 mEq buffering potential. Essentially, all excess acids must be buffered at the expense of bone-buffering reserves. If not replenished, the loss of buffering causes a slow, persistent loss of bone mineral matrix. This accelerates osteopenia and osteoporosis complication risks.

Just how low is our intake of alkaline precursors? Only 15% of the total United States population meets the fruit (2-4 servings) and vegetable (3-5 servings) recommended intakes on a daily basis.[54] Among children, only 7% consume two fruit servings and three vegetables per day, with french fries accounting for nearly 25% of vegetables, in diets of the children surveyed.[55]

Furthermore, an average North American teenager consumes three to six "cola" beverages a day. This additional daily consumption of 192-384 mEq of phosphoric acid (64 mEq per can of soda, times three to six per day) further accelerates bone loss in the young. It takes years for the cumulative load to present with clinically significant complications. However, it is likely that in coming decades we will see an acceleration of bone loss, and loss of body buffering competencies, in younger and younger people.

Acidosis and Osteoporosis

Support for the hypothesis that compensated metabolic acidosis is a foundational cause of osteoporosis comes from many sources. For some time, epidemiological studies have suggested the link between osteoporosis and animal protein intake.[68] More recently, analyses of cross-cultural fracture rates document the link between the consumption of animal protein and the incidence of hip fracture worldwide.[69] In addition, new studies report that those who consume more fruits and vegetables have higher bone mineral density than those consuming fewer of these "Alkaline Way®" foods.[70,71] Also of note

are new studies showing a three- to five-fold increase in fractures among teenage girls who regularly consume acidic soft drink beverages.[72,73]

Bone Loss Through Buffering Excess Metabolic Acids

In a study of vegetarian and animal protein diets, it was found that urinary pH was more acidic (6.17 vs. 6.55), net acid excretion was 27 mEq/day higher, and daily urinary calcium excretion was 47mg higher in those consuming animal protein. This was in spite of the fact that the diets contained the same amounts of total protein, phosphorus, sodium, potassium, and calcium. The animal protein diet, however, contained 6.8 mmol more sulfate.[56] In another study among the elderly, calcium balance was positive (+40mg/day) on a low-protein diet of 0.8 grams of protein per kilogram of body weight (56g/day for a 70 kg adult). In contrast, calcium balance was negative (-64mg/day) on a high-protein diet of 1.2 grams of protein per kilogram of body weight (84g/day for a 70 kg adult).[57] Higher protein intake would lead to even greater losses of calcium, magnesium, and other minerals.

A 50 or 60mg daily loss of calcium might not seem like much. However, over 20 years, a daily 50mg loss of calcium would translate into depletion of 365 grams of calcium, which is one-half of the average female skeletal calcium and one-third of the male's.[58] Indeed, it is not uncommon for women to lose half of their bone mass, and men, a third of their bone mass, during their lifetimes. Thus, it is possible to explain the induction of osteoporosis in our population from the causes cited above.

What is clinically underappreciated is that such loss is avoidable, witnessed by the fact that osteoporosis is uncommon in many cultures. For example, even after careful study, no sign of bone loss can be found among the Maya Indians, who eat an alkaline-rich diet.[59] Africans have been classified as "almost immune" to osteoporotic fracture.[60,61] The Chinese were found to have only one-fifth the U.S. fracture rate, despite eating as much, or nearly as much protein (but predominantly from plant sources).[62] Societies with the lowest osteoporosis rates follow the suggestions for risk reduction contained in this article. All in all, the osteoporotic fracture rate varies some 30-fold around the world.[63] This is almost entirely explainable by diet and lifestyle choices.

Clinical Implications

The contemporary Western diet leads to chronic, low-level acidosis, to the detriment of basic health and well-being. Of particular interest here is that this acidosis first forces the loss of alkalizing sodium and potassium, and then carbonate, calcium, magnesium, and other minerals from bone stores. These losses lead to excessive bone weakness, osteopenia, and osteoporosis. The solution to this problem lies in a return to a sustainable diet, rich in alkaline precursors. Consistent, weight-bearing exercise is also essential. Guidelines for development of such an "Alkaline Way® diet" are as follows:

1. The bulk of the diet should be alkalizing vegetables, fruits, lentil/pulses, nuts, seeds and spices. The chart on p.12 illustrates the effects of foods on internal

acid/alkaline balance. Sixty to 80% of foods eaten should be from the alkaline side.

2. Limit animal flesh to four ounces per day, and restrict total protein intake to 50 to 60 grams per day.
3. Maintain a fat intake of no more than 15 to 20% of total calorie intake.
4. Drink 64 ounces of high mineral (highly dissolved solids) spring water daily.
5. Fresh vegetable juice is an exceptionally good source of buffering minerals. Those with persistent low-grade acidosis might drink 2-3 eight-ounce glasses a day. We calculate that 16 to 24 ounces of juice from organic vegetables would be sufficient to correct for 40 to 50 mEq of excess organic acid.
6. Use alkalizing nutritional supplements, such as bioavailable, ionized minerals and a high quality, antigen-free ascorbate buffered with calcium, magnesium, zinc, and potassium. Add L-glutamine with pyridoxal alphaketoglutarate (PAK), Krebs' salts, cesium, rhubidium, and sesame/flax seeds as needed to keep a healthy first-morning urine pH.
7. Modify the diet and supplement sufficiently to obtain and maintain a first-morning urine pH of 6.5 to 7.5, which may reflect the existence of adequate buffering mineral reserves.
8. Reverse learned patterns of distress (sympathetic hyperactivity) by practicing relaxation responses, enjoyable activities, and weight-bearing exercise.
9. Restore tolerance to the immune defense and repair systems.

Final Thoughts

Maintenance of healthy bone and cellular pH over a lifetime is a matter of small, yet deeply important choices. Half of all older women and 20% or more of older men will suffer fractures that can, in large measure, be avoided by following the suggestions in this article. Substantial improvement in quality of life, and reduced treatment costs of $14 to $60 billion per year, are some of the benefits to be harvested.

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REFERENCES

1. Kiberistis P, *et al*: Bone health in the balance. *Science* 289:1497, 2000.
2. Dacy P, *et al*: The osteoblast: a sophisticated fibroblast under central surveillance. *Science* 289:1501-1504, 2000.
3. Teitelbaum S: Bone resorption by osteoclasts. *Science* 289:1505-1508, 2000.
4. National Osteoporosis Foundation: Osteoporosis, Fast Facts. http://www.nof.org/osteoporosis/stats.htm, April 13, 2000.
5. Ibid.
6. Wachman A, *et al*: Diet and osteoporosis. *The Lancet* 1:958-959, 1968.
7. Lemann J Jr, Lennon EJ: Role of diet, gastrointestinal tract, and bone in acid-base homeostasis. *Kidney Int* 1:275-279, 1972.
8. Barzel US, Massey LK: Excess dietary protein can adversely affect bone. *J Nutr* 128:1051-1053, 1998.
9. Anand C, Linkswiler H: Effect of protein intake on calcium balance of young men given 500mg of calcium daily. *J Nutr* 104:695-700, 1974.
10. Chu J, *et al*: Studies in calcium metabolism II. Effects of low calcium and variable protein intake on human calcium metabolism. *Am J Clin Nutr* 28:1028-1035, 1975.
11. Johnson NE, *et al*: Effect of protein intake on urinary and fecal calcium and calcium retention of young adult males. *J Nutr* 100:1425-1430, 1970.
12. Margen S, *et al*: The calciuretic effect of dietary protein. *Am J Clin Nutr* 27:548-588, 1974.
13. Schwartz R, *et al*: Effect of magnesium and protein level on calcium balance. *Am J Clin Nutr* 26:519-523, 1973.
14. Walker Rm, Linkswiler HM: Calcium retention in the adult human male as affected by protein intake. *J Nutr* 102:1297-1302, 1972.
15. Heaney RP, Recker RR: Effects of nitrogen, phosphorus, and caffeine on calcium balance in women. *J Lab Clin Med* 99:46-55, 1982.
16. Bushinsky DA: Metabolic alkalosis decreases bone calcium efflux by suppressing osteoclasts and stimulating osteoblasts. *Am J Physiol* 271:F216-F22, 1996.
17. Heaney RP: Excess dietary protein may not adversely affect bone. *J Nutr.* 128:1054-1057, 1998.
18. Kleinman JG, Lemann J Jr: Acid production. In: Maxwell MH, *et al* (eds): *Clinical Disorders of Fluid and Electrolyte Metabolism, 4th Edition.* New York: McGraw Hill, 1987, pp. 159-173.
19. Green J, Kleeman CR: The role of bone in the regulation of systemic acid base balance. *Contrib Nephrol* 91:61-76, 1991.
20. Ibid.
21. Remer T, Manz F: Estimation of the renal net acid excretion by adults consuming diets containing variable amounts of protein. *Am J Clin Nutr* 59:1356-1361, 1994.
22. Green, op. cit.
23. Remer, op.cit.
24. Ibid.
25. Frassetto L, Sebastian A: Age and systemic acid-base equilibrium: analysis of published data. *J Gerontol* 51:B91-B99, 1996.

26. Lennon EJ, *et al*: The effect of diet and stool composition on the net external acid balance of normal subjects. *J Clin Invest* 45:1601-1607, 1966.
27. Kurtz I, *et al*: Effect of diet on plasma acid-base composition in normal humans. *Kidney Int* 24:670-680, 1983.
28. Schwarz WB, Relman AS: A critique of the parameters used in the evaluation of acid-base disorders. *New Engl J Med* 268:1382, 1963.
29. Szent-Gyrogy A: *Quantum Biochemistry.* New York: Blackwell, 1957.
30. Jaffe R: Autoimmunity: clinical relevance of biological response modifers in diagnosis, treatment, and testing. *Int J Integr Med* 2(2):7-14, March/April 2000.
31. Ibid.
32. Ibid.
33. Sebastian A: Improved mineral balance and skeletal metabolism in post-menopausal women treated with potassium bicarbonate. *N Engl J Med* 330:1776-1781, 1994.
34. Ibid.
35. Frassetto L, *et al*: Potassium bicarbonate reduces urinary nitrogen excretion in postmenopausal women. *J Clin Endocrinol Metab* 82:254-259, 1997.
36. Ibid.
37. Halperin ML, Goldstein MB: *Fluid, Electrolyte, and Acid-Base Physiology: A Problem-Based Approach, 3rd Edition.* Philadelphia: WB Saunders Company, 1999.
38. Ibid.
39. Green, op. cit.
40. Cohn SH, *et al*: Body elemental composition: comparison between black and white adults. *Am J Physiol* 232:E419-E422, 1977.
41. Armstrong WD, Singer L: Composition and constitution of the mineral phase of bone. *Clin Orthop* 38:179-190, 1965.
42. Flynn MA, *et al*: Total body potassium in aging humans: a longitudinal study. *Am J Clin Nutr* 50:713-717, 1989.
43. Poyart CF, *et al*: The bone CO2 compartment: evidence for a bicarbonate pool. *Respir Physiol* 25:89-99, 1975.
44. Vatassery GT, *et al*: Determination of hydroxyl content of calcified tissue mineral. *Calcif Tissue Res* 5:183-188, 1970.
45. Green, op. cit.
46. Triffitt JT, *et al*: A comparative study of the exchange in vivo of major constituents of bone mineral. *Calcif Tissue Res* 2:165-176, 1968.
47. Green, op. cit.
48. Schwarz, 1963, op. cit.
49. Remer, op. cit.
50. Frassetto, 1996, op. cit.
51. Remer, op. cit.
52. Barzel, op. cit.
53. Halperin, op. cit.
54. Schweitzer C, *et al:* Dietary intake of carotenoids, fruits, and vegetables in the US: CSFII 1994-1996, a national survey. Proceedings from the 12th International Carotenoid Symposium, July 18-23, 1999, Cairns, Australia.
55. Krebs-Smith SM, *et al*: Fruit and vegetable intakes of children and adolescents in the United States. *Arch Pediatr Adolesc Med* 150:81-86, 1996.

56. Breslau NA, *et al*: Relationship of animal protein-rich diet to kidney stone formation and calcium metabolism. *J Clin Endocrinol Metab* 66:140-146,1988.
57. Licata AA, *et al:* Acute effects of dietary protein on calcium metabolism in patients with osteoporosis. *J Gerontol* 36:14-19, 1981.
58. Barzel, op. cit.
59. Beyene Y: Cultural significance and physiological manifestation of menopause: a biocultural analysis. *Cult Med Psychiatry 10:47-71, 1986.*
60. Luyken R, Luyken-Koning R: Studies on the physiology of nutrition in Surinam VIII. Metabolism of calcium. *Trop Geogr Med* 13:46-54, 1961.
61. Chalmers J, Ho K: Geographical variations in senile osteoporosis. *J Bone and Joint Surgery* 52B:667-675, 1970.
62. Campbell TC, Cox C: *The China Project.* Ithaca: New Century Nutrition, 1996.
63. Melton L, Riggs B: Epidemiology of age-related fractures. In: *The Osteoporotic Syndrome: Detection, Prevention and Treatment.* New York: Grune and Stratton, 1983.
64. Krieger NS, et al: Acidosis inhibits osteoblastic and stimulates osteoclastic activity in vitro. *Am J Physiol* 31:F442-F448, 1992.
65. Arnett TR, Spowage M: Modulation of the resorptive activity of rat osteoclasts by small changes in extracellular pH near the physiological range. *Bone* 18:277-279, 1996.
66. Arnett TR, Dempster DW: Effect of pH on bone resorption by rat osteoclasts in vitro. *Endocrinology* 119:119-124, 1986.
67. Grinspoon SK, *et al:* Decreased bone formation and increased mineral dissolution during acute fasting in young women. *J Clin Endocrinol Metab* 80:3628-3633, 1995.
68. Marsh A, *et al:* Cortical bone density of adult lactovegetarian women and omnivorous women. *J Amer Diet Assoc* 76:148-151, 1980.
69. Abelow BJ, *et al*: Cross-cultural association between dietary animal protein and hip fracture: a hypothesis. *Calcif Tissue Int* 50:14-18, 1992.
70. Tucker, *et al*: Potassium, magnesium, and fruit and vegetable intakes are associated with greater bone mineral density in elderly men and women. *Am J Clin Nutr* 69:727-736, 1999.
71. New SA, *et al*: Dietary influences on bone mass and bone metabolism: further evidence of a positive link between fruit and vegetable consumption and bone health? *Am J Clin Nutr* 71:142-151, 2000.
72. Wyshak G, Frisch RE: Carbonated beverages, dietary calcium, the dietary calcium/phosphorous ratio, and bone fractures in girls and boys. *J Adolesc Health* 15:210-215, 1994.
73. Wyshak G: Teenaged girls, carbonated beverage consumption, and bone fractures. *Arch Pediatr Adolesc Med* 154:610-613, 2000.

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